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=> d his

     (FILE 'HOME' ENTERED AT 16:24:32 ON 21 JAN 2009)

     FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH, LIFESCI' ENTERED AT 16:24:58 ON
     21 JAN 2009
L1           389 S (INCREAS? OR ENHANC?)(5A)(NEURAL OR NEURON?)(4A)(TRANSPORT OR
L2         15084 S BDNO OR NT-4 OR GDNF
L3         12151 S BDNF PR NT-4 OR GDNF
L4             2 S L1 AND L3
L5             2 DUP REM L4 (0 DUPLICATES REMOVED)

=> d bib ab 1-2 l5

L5   ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2009 ACS on STN
AN   2003:788860  CAPLUS
DN   140:71459
TI   GDNF increases the survival of developing oculomotor neurons
     through a target-derived mechanism
AU   Chen, Jennifer; Butowt, Rafal; Rind, Howard B.; von Bartheld, Christopher
     S.
CS   MS 352, Department of Physiology and Cell Biology, University of Nevada
     School of Medicine, Reno, NV, 89557, USA
SO   Molecular and Cellular Neuroscience (2003), 24(1), 41-56
     CODEN: MOCNED; ISSN: 1044-7431
PB   Elsevier Science
DT   Journal
LA   English
AB   Glial cell line-derived neurotrophic factor (GDNF) is the most
     potent motoneuron survival factor.  The authors show here that in the
     chick oculomotor system, endogenous GDNF is derived largely from
     extraocular muscle but less from glial cells and not from muscle spindles.
     Increased levels of GDNF exclusively in the target rescued 30%
     of oculomotor neurons that would normally die during developmental cell
     death, a rate of rescue similar to that with systemic GDNF
     application.  Thus, GDNF supports motoneuron survival in a
     retrograde, target-derived fashion, as opposed to a local paracrine route
     or an indirect route via sensory afferents.  Persephin, another member of
     the GDNF family, did not increase survival with target delivery,
     despite its retrograde transport from the target.  Unlike GDNF,
     however, persephin increased neurite outgrowth from oculomotor nuclei in
     vitro.  Thus, one GDNF family member acts as a muscle-derived
     retrograde survival factor, whereas another one has distinct functions on
     neurite outgrowth.
RE.CNT  68    THERE ARE 68 CITED REFERENCES AVAILABLE FOR THIS RECORD
              ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5   ANSWER 2 OF 2  CAPLUS  COPYRIGHT 2009 ACS on STN
AN   2002:520318  CAPLUS
DN   137:211245
TI   Lentivirally delivered glial cell line-derived neurotrophic factor
     increases the number of striatal dopaminergic neurons in primate models of
     nigrostriatal degeneration
AU   Palfi, Stephane; Leventhal, Liza; Chu, Yaping; Ma, Shuang Y.; Emborg,
     Marina; Bakay, Roy; Deglon, Nicole; Hantraye, Philippe; Aebischer,
     Patrick; Kordower, Jeffrey H.
CS   Department of Neurological Sciences, Rush-Presbyterian-St. Luke's Medical
     Center, Chicago, IL, 60612, USA
SO   Journal of Neuroscience (2002), 22(12), 4942-4954
     CODEN: JNRSDS; ISSN: 0270-6474
PB   Society for Neuroscience
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DT Journal
LA English
AB The primate striatum contains tyrosine hydroxylase (TH)-immunoreactive (ir) neurons, the nos. of which are augmented after dopamine depletion. Glial cell line-derived neurotrophic factor (GDNF) strongly modulates the viability and phenotypic expression of dopamine ventral mesencephalic neurons. The effect of GDNF on TH-ir neurons intrinsic to the striatum has yet to be investigated. In the present study, stereol. counts of TH-ir striatal neurons in aged and parkinsonian nonhuman primates revealed that GDNF delivered via a lentiviral vector (lenti-) further increased the number of these cells. Aged monkeys treated with lenti-GDNF displayed an 8-fold increase in TH-ir neurons relative to lenti-β-galactosidase-treated monkeys. Unilateral 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine treatment alone in young monkeys resulted in a bilateral 8-fold increase in TH-ir striatal cells. This effect was further magnified 7-fold on the side of lenti-GDNF treatment. These cells colocalized with the neuronal marker neuronal-specific nuclear protein. Some of these cells colocalized with GDNF-ir, indicating that an alteration in phenotype may occur by the direct actions of this trophic factor. Thus, GDNF may mediate plasticity in the dopamine-depleted primate brain, which may serve to compensate for cell loss by converting striatal neurons to a dopaminergic phenotype.
RE.CNT 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s tetanus(3a)toxin
L6 13457 TETANUS(3A) TOXIN

=> s l5 and l6
L7 0 L5 AND L6

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